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SWIM-BLADDER ECTASIA IN THE TREVALLY CARANX GEORGIANUS

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ABSTRACT

All of 13 C. georgianus 470 to 540 mm in fork length caught off southeast Tasmania had cavities 2 to 20 mm across in the anterior musculature over the vertebral column. The cavities were lined with smooth muscle and connective tissue, and filled with gas. Cultures for bacteria were negative. No helminths were present, and histologically there was no sign of any protozoan infection or any inflammation. All the cavities on one side of a fish connected to a horizontal chamber up to 200 mm long that ran immediately dorsolateral to the vertebral column, and opened at the anterior end into the swim bladder via a small duct. A similar but not identical system of tubes was present on the other side of the fish. It is suggested that over a period of time pressure in the swim bladder had stretched the bladder wall and forced it to swell into the tissues. The cavities in the musculature reduced the marketability of the fish.

INTRODUCTION

Silver trevally, Caranx georgianus, are caught by line and net fishermen in southern Australian waters, particularly in the summertime. A series of good catches in early 1981 focused attention on what were apparently holes in the musculature.

Though their existence in trevally has been known for many years, on this occasion processing companies refused to accept fish with these 'cancerous' abnormalities. The present study was therefore undertaken to determine their origin.

Possible causes that were considered were: bacteria, as Aeromonas salmonicida and Renibacterium salmoninarum in other fishes can cause breakdown of the musculature to produce discrete pus-filled cavities; protozoa, such as Kodoa clupeidae; and parasitic helminths, as larval trypanorhynch tapeworms sometimes produce a bladder-like growth that may be found in the musculature.

MATERIALS AND METHODS

Thirteen fish, 470 to 540 mm in fork length, and one fish 180 mm long, caught off southeast Tasmania in February 1981, were dissected within 48 hours of being caught. Four other fish, 420 to 480 mm long and caught at the same time were used in making casts. Four fish 440 to 470 mm long, caught in the same area, were dissected fresh in May, 1978, and one formalin-fixed fish, 340 mm in length and caught in the Tamar River, northern Tasmania, in February, 1975, was also examined.

Attempts to culture bacteria were made on agar plates using the following media; Trypticase Soy Agar, Columbia Blood Agar, 0.1 Columbia Blood Agar, and Mueller-Hinton with cocarboxylase. Inoculated media were incubated at 22° C for 5 days. Tissues were fixed in formalin and Bouin's fixatives, and stained with haematoxylin and eosin, Giemsa, and Verhoeff's elastin stain.

Fish to be injected with epoxy resin were held ventral side uppermost, the body cavity opened, an incision made in the swim bladder, and the resin introduced using a 20 ml syringe. In two of the fish, prior to injection, part of the musculature was removed to expose the ends of some of the canals, so that the resin could pass from the swim bladder through the canals to the outside, thus ensuring that the full length of the canal was filled. The resin used was Epirez 133 Hydrophobic Epoxy Binder 2 part mix. It was left to harden for 2 to 3 days after which the fish tissue was macerated within a solution of hot 1 molar sodium hydroxide and the casts freed.

Fish age was determined from the number of annuli on otoliths and scales.

RESULTS

All fish dissected over 330 mm had cavities in the musculature. The cavities ranged from 2 to 20 mm in diameter and were lined by a white pliable tissue (Figure 1). They enclosed no liquid or sign of necrotic tissue, and appeared to contain only gas. Cavities were grouped in the anterior part of the musculature dorsal to the vertebral column, and were more prevalent in some fish than others.

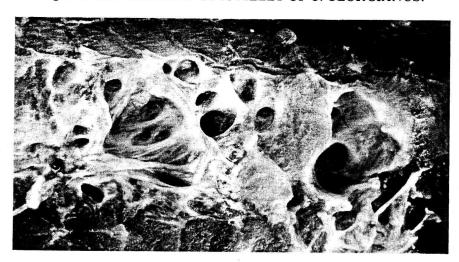


Figure 1. THE CAVITIES OF A FILLET OF C. GEORGIANUS.

In the 13 large fish dissected in 1981, the holes on one side were interconnected, and joined to a long horizontal chamber that lay immediately dorsolateral to the vertebral column (Figures 2 and 3). The length of this chamber varied between fish, and between the left and right sides of the same fish (Table 1). Finger-like projections from this chamber extended into the musculature, and it was these that appeared as holes in the fillets. The extrusions tended to run between the dorsal vertebral spines, though they did not cross the midline of the fish, and penetrated dorsally up to a muscular septum. The degree of development of the projections varied, some fish having a long horizontal chamber with only 2 projections, others having a short chamber with four or five well developed projections.

In all cases, however, at the anterior end of the chamber there was a small tube that ran ventrally around the vertebral column and opened into the swim bladder beneath. This connecting duct was between the 3rd and 4th vertebrae, or between the 4th and 5th vertebrae, sometimes being between different vertebrae on the two sides of the same fish (Figures 2 and 3).

Figure 2. THE LAYOUT OF THE EXTRUSIONS ON THE LEFT SIDE OF ONE FISH.

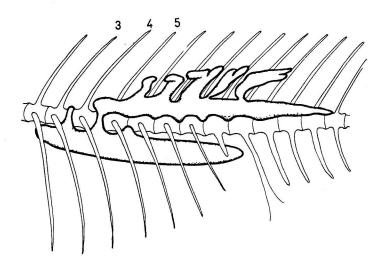
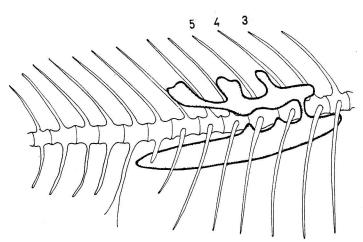


Figure 3. EXTRUSIONS ON THE RIGHT SIDE OF THE FISH SHOWN IN Fig. 2.



These connections and the shapes of the chambers were clearly demonstrated by the resin casts (Figure 4). Each swim bladder contained several dorsal pockets where it fitted between the ribs. Evidently one of these on each side of the fish had broken through into the tissue above. Two of the four casts showed a feature that had been overlooked in the dissections, a pair of horizontal chambers ventral to the vertebral column, running from the posterior of the swim bladder into the caudal musculature. These also had lateral projections though they were less well developed than those in the dorsal musculature.

Histologically, the cavities were lined with a thin mucosa and a mixed layer of smooth muscle and connective tissue. Behind these was an area of fat cells within which were thick sheets of collagen fibres. The fat cells were in contact with striated muscle fibres, some of which showed hyaline degeneration. There was no sign of inflammation, and no evidence of any bacterial, protozoal or helminth infection. The wall of the swim bladder proper also contained smooth muscle and connective tissue though these formed two separate layers, the tunica muscularis and the tunica serosa. Fat cells were absent.

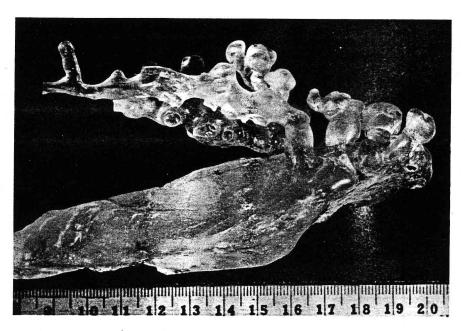
No bacteria were cultured on the plates.

The 180 mm fish dissected was free of any swim bladder extrusions. Five of the fish 490 to 520 mm long were determined to be 7+ years old, whilst the 180 mm fish was 2+years old.

Table 1. THE LENGTHS OF THE HORIZONTAL CHAMBERS IN TEN C. GEORGIANUS.

| Fish length | left side mm | right side mm |
|-------------|--------------|------------------|
| 470 | 130 | 75 |
| 480 | 45 | 55 |
| 490 | 85 | 110 |
| 490 | 190 | 40 |
| 500 | 200 | 75 |
| 500 | 42 | 40 |
| 520 | 160 | 80 |
| 520 | 120 | 130 |
| 540 | 160 | 78 |
| 550 | 110 | 80 |
| | | |

Figure 4. A RESIN CAST FROM ONE FISH SHOWING THE SWIM BLADDER AND ITS EXTRUSIONS. ANTERIOR OF FISH TO RIGHT.



DISCUSSION

The anomaly was evidently not the result of any bacterial or parasitic infection. Proliferation of the swim bladder as a result of a swim bladder tumour has been reported in three species of fish.^{2,4,5} However, in these cases the tumours were solid masses of tissue quite unlike the gas filled spaces seen here. Finger-like projections are a normal feature of the swim bladder in some other families, particularly sciaenids.⁶ However they have not been reported from carangids.

For several reasons, the trevally holes are thought to be abnormal. They are highly irregular in number and form in fish of the same size, and they develop differently on the two sides of individual fish. The fat cells surrounding the cavities suggest that muscles in the area have broken down, as adipose tissue is sometimes the product of muscle damage. Extrusions of the swim bladder have not been noticed in *C. georgianus* in New Zealand waters (B. Webb, personal communication).

The condition superficially resembles 'pneumothorax' of mammals. However, the gas had not been forced out of the swim bladder. Rather it was in the tissues still surrounded by an extension of the bladder wall. This integrity plus the lack of inflammation indicate that the condition had developed gradually, possibly as a result of a series of high pressures in the bladder. Increased pressure such as would result from a rapid change in depth would normally be compensated for by changes in bladder size, unless either a part of the wall was weakened in some way, or the bulk of the wall had lost some of its elasticity. This particular population of *C. georgianus* is prone to another abnormal development, osteomas on various pterygiophore and haemal spines.³ The metabolic malfunction that results in the deposition of these masses may in some way be linked to the swim bladder extrusions described here.

Since completing this work the senior author has found swim bladder ectasia in an 88 cm snapper *Chrysophyrys unicolor* from the Spencer Gulf, South Australia. The extrusions were demonstrated by x-ray photography and confirmed by dissection. Verbal reports indicate that 'holes' are not uncommon in the flesh of snapper in southern Australian waters.

That the same syndrome occurs in two unrelated species of fish raises the possibility that it is triggered by some environmental factor. A low pressure wave, for example, may cause the initial extrusion, which with time then progresses through the tissues as a result of the fish's normal changes in depth. Low pressure waves of suitable wavelengths are produced by some imploding devices used in seismic surveys and by cavitation of the propellers of large tugs and ocean-going vessels. However, if low pressure waves are the cause, one would expect swim bladder ectasia to be a world-wide phenomenon. As far as we can determine it is not, for there are no other reports of it in the literature. Until more information is available, we have to conclude that it is probably caused by a genetic abnormality in the fish populations concerned.

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